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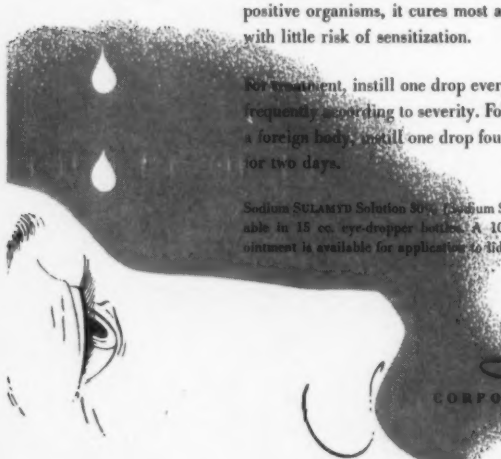

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Vol. VII *June 1951* No. 7

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THE ROLE OF TRACHEOTOMY IN THE MANAGEMENT OF BULBAR POLIOMYELITIS

Report of Two Cases*

Allan A. Walker, M.D.
Antonio Busquets, M.D.
Joseph M. Lo Presti, M.D.

INTRODUCTION

The following two case reports are presented to emphasize to the medical practitioner that a tracheotomy in carefully selected patients with bulbar poliomyelitis may be life saving.

CASE REPORTS

Case 1. W. T., a seven year old colored boy was admitted to Children's Hospital on October 10, 1950. Two days prior to admission, he developed malaise, anorexia and urinary frequency. On the day prior to admission he developed fever and regurgitated fluids through the nose. On the morning of admission he had been unable to swallow and his speech had become slurred. The child was toxic, restless, and overactive. The spoken voice was forced. The neck was moderately stiff. The flexors of the neck were completely paralysed. He was unable to swallow his saliva. Respiratory rate was 38 and regular. There was a right facial paralysis. The admission temperature was 105° rectally. Spinal fluid contained nine cells with normal sugar and protein content. On October 11, his condition became critical. He became cyanotic and developed paradoxical respirations. Inspiration was noisy, apparently due to fixation of the cords in the mid-line. Tracheotomy was performed under local anaethesia. On October 12, 1950 the right diaphragm became paralysed and the respirations became quite shallow and irregular and feeble and he was placed in a respirator. He improved slowly. On October 28 direct laryngoscopy revealed complete paralysis of the right vocal cord and about 20 per cent motion of the left cord. On November 27 the patient was able to swallow and the laryngeal paralysis had disappeared. The tracheotomy tube was removed without trouble on this date. The patient was discharged December 24, 1950 to the Out Patient Department for treatment of residual weakness of the anterior cervical muscles.

Case 2. T. T., an eleven year old white boy was admitted to Children's Hospital on October 9, 1950. Two days prior to admission he had developed fever, malaise, inability to swallow and escape of fluids from the nose. Physical examination revealed a toxic child with a nasal twang to the voice, moderate nuchal rigidity, moderate stiffness of the back, absent pharyngeal reflex, and inability to swallow. Temperature: 103° rectally, respiration: 22 to 25/min., blood pressure: 126/68. Spinal fluid contained 129 cells with a normal sugar and protein. He was placed in Trendelenberg position, given constant pharyngeal aspirations and parenteral fluids. He became progressively worse, with marked respiratory embarrassment, involvement of the intercostal muscles, cyanosis and extreme restlessness, irrationality, and irritability.

* These two cases were admitted to the ward service of Edward Lewis, M.D.

His blood pressure rose steadily, reaching 150/106. On October 11, 1950 a tracheotomy was performed under local anaesthesia and the patient was put in a respirator. His condition improved promptly. On the next day he was responsive. The blood pressure dropped to 120/80. On October 17, 1950 he was able to spend long intervals out of the respirator. On October 23, 1950 the patient was able to swallow and to cough. The tracheotomy tube was removed on this date. The patient was discharged on November 22, 1950 to the Out Patient Department of Physiotherapy for residual weakness of the shoulder muscles.

DISCUSSION

Allan A. Walker, M.D.: Within the past several years there has been an increasing recognition of the value of tracheotomies in selected cases of poliomyelitis. Galloway[1] in his articles dealing with this problem has emphasized the effects of anoxia in these patients. He states that the symptoms of anoxia may be ascribed to sepsis, decompensation, shock and virus infection. The mental symptoms vary from early exhilaration to depression, confusion, irrationality, lethary and coma. Restlessness and dyspnea are usually early signs and cyanosis is usually a late sign.

Sjoberg[2] in discussing the indications for interference in the management of suffocation in spinobulbar poliomyelitis classifies the lesions into five groups:

(a) Peripheral respiratory paresis with secretional retention.

(b) Aspiration in bulbar cranial nerve lesions.

(c) Bilateral paralysis of the paryngeal abductors combined with bilateral paresis of the phargnx.

(d) Spinobulbar lesions in which the non-paralysed muscles draw the hyoid bone upward and backward and strangle the patient.

(e) The prophylactic group in which operative treatment is performed early in anticipation of anoxia.

Stobel and Canfield[3] emphasize the fact that poliomyelitis may interfere with the mechanisms of coughing and swallowing so the patients can not maintain clear airways. They feel that therapeutic tracheotomy should be performed on any respirator patient who exhibits signs of bulbar involvement, or who displays persistent cyanosis or struggling and in any non-respirator patient who is unable to keep his airway free of secretion.

The two cases presented illustrate the value of tracheotomy in bulbar poliomyelitis. The first boy had a combined pharyngeal and laryngeal abductor paralysis, forming an anatomical situation incompatible with survival. The second case illustrates the effects of anoxia on the systems, as evidenced by irrationality, cyanosis, respiratory embarrassment and impending circulatory collapse.

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Joseph M. Lo Presti, M.D.: The mortality rate of acute anterior poliomyelitis varies between 5 and 10%. However, over 90% of these deaths are due to bulbar involvement. Bulbar poliomyelitis, therefore, is the most important and serious form that this disease assumes. Based on the clinical observations of Baker[1] and substantiated by the pathological findings reported by Bodian[2], bulbar poliomyelitis has been classified into five main categories. Only on rare occasions does one of these groups exist by itself. Most frequently there is a dove-tailing of groups with one predominating. Over half of the patients with bulbar poliomyelitis will have cranial nerve involvement:

1. *The upper cranial nerve group.* This includes involvement of the first eight cranial nerves. The cardinal clinical features are ptosis of the eyelid, anisochorea, facial paralysis, and strabismus. By itself, such involvement is not dangerous. However, it should make the practitioner wary of the possibility of further involvement of more vital centers.

2. *The lower cranial nerve group.* This includes involvement of the 8th, 9th, 10th, 11th, and 12th cranial nerves. By far, the nerve most frequently involved is the 10th (vagus). Lower cranial nerve involvement is the type most commonly encountered. This group is definitely hazardous to the life of the patient for three reasons:

   a. The accumulation of secretions or vomitus in the posterior pharynx, with the danger of aspiration.
   b. Closure of the glottis in reflex spasm.
   c. Abductor paralysis of the vocal cords with their approximation in the mid-line.

The clinical symptomatology in this group is a nasal twang to the voice, hoarseness, the accumulation of oropharyngeal secretions, and the regurgitation of fluids through the nose.

3. *Respiratory center group.* Most of these patients show some cranial nerve involvement. The outstanding symptoms are respiratory, characterized by the development of irregularities of rhythm and depth of respirations. The respirations tend to be shallow and there are prolonged intervals between inspirations. *These respiratory symptoms occur despite strong intercostal muscles and diaphragm.* The patient dies of progressive respiratory failure.

4. *The circulatory center group.* Such involvement is most frequently observed in the most severe cases of bulbar poliomyelitis in which the cranial nerve nuclei and the respiratory center are predominately affected.

When the symptoms of circulatory involvement supervene in any form of bulbar poliomyelitis, the prognosis is extremely grave. The patients have a dusky red, flushed, florid appearance. The lips are a deep cherry red. The pulse is very rapid and irregular, ranging between 150 and 200. The patient dies in peripheral circulatory collapse.

5. *The encephalitic group.* Encephalitic symptoms occur commonly in bulbar poliomyelitis and appear within the first few days of the illness. Whether these symptoms are secondary to cerebral hypoxia or to direct involvement of the brain by the virus is an unestablished and debatable point.

The treatment of bulbar poliomyelitis in the acute stage is primarily a nursing problem. If the patient can be maintained through the acute phase of the illness, the prognosis is favorable. In general, bulbar poliomyelitis patients *do not do well in the respirator*. The respirator is life saving in the group of patients who have high spinal cord involvement with paralysis of the intercostal muscles and/or the diaphragm. There are two reasons why the respirator is contraindicated in the management of bulbar poliomyelitis:

1. With each exertión of negative pressure over the thoracic cage the accumulation of oropharyngeal secretions will be suctioned into the bronchial tree.

2. The use of the respirator presupposes central control of respirations. The patient with bulbar poliomyelitis who has respiratory embarrassment has partially lost this central control. He is therefore, incapable of accommodating to the respirator, and a fatal termination will be hastened.

Wilson[3] has outlined the recommended treatment of the majority of patients with bulbar poliomyelitis. There are six essential features in this management:

1. *The patient is placed in Trendelenberg position* in order to utilize gravity as an aid in draining the oropharynx. These patients should not be permitted to lie in the supine position. They should be kept on their sides or in the prone position.

2. *Careful, gentle suction of the oropharynx whenever necessary.* For this purpose it is best to utilize a soft rubber catheter to avoid traumatizing the posterior pharyngeal wall. Suction should never be performed through the nose. These patients already have respiratory embarrassment and passing a tube through the nares only will serve to cause a rapid edema of the nasal turbinates.

3. *The administration of oxygen,* preferably by means of a nasal catheter. An oxygen tent for this purpose is impractical because it obscures the careful observation which is mandatory in the management of bulbar patients.

4. The patients *should not be permitted to take anything by mouth.* They have difficulty in swallowing and the danger of vomiting or aspiration is constantly present during the acute stage of the illness. Gavage should not be attempted at this time.

5. *The daily administration of parenteral pencillin* is recommended as a prophylactic measure against secondary invaders.

6. *The maintainance of electrolyte and fluid balance.* It is particularly important that these patients receive an adequate, daily intake of protein. This is accomplished best by the parenteral administration of whole blood or plasma each day.

The role of tracheotomy in the management of patients with bulbar poliomyelitis is a much discussed and controversial problem. However, most authorities agree that there are definite, specific indications for tracheotomy. There are, perhaps, four groups of patients in whom tracheotomies are indicated:

1. *If adequate and trained nursing personnel is not available* for the management of these patients, the mortality rate will be lowered if tracheotomies are routinely performed. It is much easier from the nursing standpoint to handle a tracheotomized patient.

2. Some patients with bulbar poliomyelitis are so apprehensive that they *fail to comprehend or cooperate.* At times, a tracheotomy in such cases will simplify the management.

3. A small number of bulbar patients will have *abductor paralysis of the vocal cords or the glottis will be closed in reflex spasm.* Obviously, a tracheotomy will be life saving in this small group.

4. Another small percentage of bulbar patients will have *comcomitant involvement of the high spinal thoracic type.* Because of intercostal muscle and/or diaphragm paralysis this group will be candidates for respirator care. Their management will be simplified, if, prior to being placed in a respirator, a tracheotomy is performed.

There are two large categories who have respiratory embarrassment in poliomyelitis. The first group includes bulbar patients who have involvement of the respiratory center and the second embraces those who have high spinal thoracic involvement. It is essential that these two be differentiated since their management is entirely different. Although the respirator is life saving in the group with intercostal and/or diaphragm paralysis, it will only hasten the end in those patients who have respiratory center involvement. From the clinical standpoint, the simplest way to differentiate the two is the respiratory rate. When respiratory center paralysis is the basis for respiratory difficulty, *the respirations are grossly irregular.* On the other hand, when the respiratory embarrassment is due to high spinal paralysis, *the respirations, though rapid, remain regular.*

The treatment of bulbar poliomyelitis patients with involvement of the respiratory center is a problem. Sarnoff[4] has recently developed an electrophrenic stimulator. A stimulating electrode is placed over the phrenic nerve. Electrical impulses are sent down the nerve and cause contraction of the diaphragm. At the same time, impulses are transmitted up the nerve and inhibit any impulses originating from the center. In effect, a substitution respiratory center is produced by electrophrenic stimulation. It would be premature to state that electrophrenic stimulation is the answer to the management of these patients, but it is conceivable that such will prove to be the case. Further trial is necessary for adequate evaluation.

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A REPORT OF A CASE OF ACRODYNIA TREATED WITH BAL

Case Report No. 209

Gordon W. Daisley, Jr., M.D.
Hugh Clark, M.D.
Preston A. McLendon, M.D.

B. C., a three year old white female was admitted to the service of Dr. Preston A. McLendon, November 16, 1949 with complaints of burning and itching palms and soles, irritability, anorexia, and photophobia of about two months' duration.

The present illness began in the first week of September, 1949 when it was noted that she became extremely anorexic. In the following two months, she did not thrive; her hair became thin and dry, and she lost her finger nails. She began to complain of itching of her soles and palms, and her mother stroked them hourly to give relief. It was noted later that an erythematous blush appeared on her body at times, usually lasting several hours, and the itching was most intense at these times. Her skin became very dry, and roughened, particularly about the mouth and on her hands and feet. The fingers became reddened as did the feet. Vitamin preparations and liver extract were of no avail. She suffered a personality change; became restless and irritable, cried a great deal and seemed most content when she buried her head in the covers between her knees, protecting her eyes from the light.

Because of these prolonged and progressive symptoms, particularly the roughened, reddened hands and feet and the burning and itching, on November 15, 1949 a tentative diagnosis of acrodynia was made. The diagnosis was corroborated by the finding of mercury in the urine and she was admitted to receive a course of BAL therapy.

The past history revealed that on July 20, 1949, she had begun a fourteen-day course of anti-rabies shots because she had been in contact with a rabid dog. Ten days after completion of this therapy (August 16, 1949), she was seen by a physician who noted a small amount of rash on her forehead and chin; 5 per cent ammoniated mercury with zinc oxide ointment was applied, and within 72 hours the rash had disappeared. On August 24, 1949 there occurred a generalized erythematous, vesiculopapular rash, which included the palms and soles. There was an associated stomatitis, and her temperature was 102.6 F. She was given 300,000 units of penicillin and Burrow's solution soaks were begun, but the patient became progressively worse, with rising fever, intense pruritis, edema of the face, and beginning exfoliation. She was first admitted to this hospital on August 25, 1949.

Past medical history and family history contributed only that the child had hay fever, and there was a history of allergy on the paternal side of the family. Ten other persons received the anti-rabies shots at the same time as the patient but none of these suffered a reaction. Physical examination on that admission was limited to the severe eczematoid urticarial rash, and the stomatitis. Temperature was 103.0 F.

Her course in the hospital at the first admission was one of gradual recovery; she spiked daily temperatures to 103.0 F. for seven days, and she completely exfoliated. The edema became generalized and did not subside for eight days. Her weight was a constant 30 pounds. Therapy consisted of a special non-allergic diet, anti-histamines by mouth and in lotions, boric acid soaks and ointments, penicillin 300,000 units daily, ascorbic acid 300 milligrams daily, and intravenous and subcutaneous glucose in saline in support. She was gavaged for two days prior to discharge because of anorexia. Laboratory examinations on this admission revealed a hemoglobin of 13 grams with 4,300,000 red blood cells; 26,900 white blood cells, with 74 neutrophiles, 23 lymphocytes, 2 monocytes, and 1 eosinophile per 100 cells. Urinalysis was normal. On August 31, total serum proteins were recorded as 4.98 grams per cent. Culture of the oral lesions on September 1, 1949 grew many alpha streptococci, moderate numbers of *Staphylococcus aureus*, micrococci, and *Neisseria*, and a few *E. coli*. The patient was discharged on September 2, 1949 with a normal temperature, subsiding rash; and it was believed that she had suffered a serum reaction or possible ammoniated mercury intoxication. The discharge diagnosis was exfoliative dermatitis.

Physical examination on this present admission revealed a pale, thin, hypotonic, irritable little girl, weighing 27 pounds, who continually had her head in the covers. There was short-cropped, thin, coarse hair, and her finger nails were normal. The feet and hands were dry, rough, and erythematous. Systolic blood pressure was 124. There was a cheilosis of the mouth present and the front teeth were a dirty grey color. Otherwise, the findings were within normal limits.

During this hospital stay she received a 14-day course of BAL in the following dosages: 0.4 milliliters of a 10 per cent solution (approximately 3 milligrams per kilogram) every 4 hours for 48 hours, then 0.4 milliliters every 6 hours for the next 24 hours, and then every 12 hours for the remainder of the 14-day period. Daily blood pressures were recorded and the systolic pressure fell gradually to 88 from 124.

No toxic reactions to BAL developed. The temperature was elevated to 101.0 F. for 24 hours on the fifth hospital day, otherwise it remained normal. The original mercury level on a 24-hour urine specimen, prior to institution of BAL therapy, was 0.04 milligrams per 1000 milliliters. After three days of therapy, on November 20 1949, the value was found to be 0.46 milligrams per 1000 milliliters. Another on November 30, 1949, just before therapy was discontinued, was 0.08 milligrams per 1000 milliliters. All of the determinations were performed at the National Institutes of Health. (Normal value—0.004 milligrams per 1000 milliliters)

Improvement in this 14-day period was not marked, but the erythema and complaints of itching gradually disappeared, although she was irritable during her whole stay. Her appetite did not improve. By the end of a month, however, definite changes in personality and appetite were noted. A final mercury level on January 16, 1950 was 0.05 milligrams per 1000 milliliters.

A year follow-up period reveals the child to be normal, and no recurrence of symptoms has appeared. She is a cheerful, happy, and cooperative child with normal tissue and skin, and the hair is luxuriant and glossy.

DISCUSSION

This patient well satisfies the criteria established by Bilderback for the diagnosis of acrodynia,[1] for as he states, "The outstanding and distinctive symptoms are: pink hands and feet, desquamation, scarlet cheeks and tip of nose, alopecia, salivation, loss of several or all of teeth, occasionally loss of nails or phalanges, excessive perspiration, evanescent rashes, marked hypotonia, itching, burning and marked pain of the extremities, increased pulse rate and blood pressure, photophobia, insomnia, and apathy alternating with extreme irritability."

All of the above symptoms were noted in this child except the excessive perspiration. In a well developed case, it is stated that no other disease of childhood gives such marked and continuous sweating.[1] The typical facies and jackknife posture (head between the legs turned away from the light) were noted.

Warkany and Hubbard[2] first produced evidence that mercury is present in the urines of patients with acrodynia, and reported a series of 20 patients. Since that time, with physicians aware of this, the majority of cases reported have included a history of exposure to some mercurial compound. Propriety teething powers and vermifuges are particularly common offenders. In this case the application of ammoniated mercury to the face for a minimal skin rash occurred, and eight days later her first severe toxic symptoms were noted, diagnosed as exfoliative dermatitis. In retrospect, her illness in August, was probably due to ammoniated mercury intoxication.

Bivings and Lewis[3] reported successful treatment of the disease with BAL (2,3-dithiopropanol) in 1948, as did Elmore,[4] and since that time many case reports of similar success have appeared in the literature. Apparently, the earlier in the course of the disease therapy is instituted, the

more rapid are results obtained. Over two months had elapsed before BAL was given to this patient; and one month after therapy was begun, she was almost fully recovered. If untreated, the disease is self-limited, but usually runs a much longer course. Bilderback states it lasts "several months."[1] Occasionally, death occurs. It is not uncommon for the disease entity to be present three or four months before diagnosis is made. It seems evident, therefore, that the course of BAL benefited this patient materially.

The therapeutic procedure used in the case is essentially the same as recommended by Bilderback, but was maintained for 14 days rather than 10. Three milligrams per kilogram per dose was employed. The patient was watched carefully for the appearance of any shock-like state, but these side-effects such as described by Beale and Schrum[5] were not noted. (Another patient in the hospital at the same time did show a shock-like state while on BAL therapy, however.[6]) The tremendous outpouring of mercury into the urine during the course bespeaks the effectiveness of the therapy.

SUMMARY

A typical case of acrodynia is presented and the first such case treated with BAL at the Children's Hospital, Washington, D. C. is reported. A brief outline of the criteria for diagnosis, and specific therapy for the disease is discussed.

We are indebted to D. H. Byers, Senior Scientist, Laboratory Section, Division of Industrial Hygiene, National Institutes of Health, for the determinations of mercury levels in the urine.

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SUBDURAL EFFUSIONS IN PYOGENIC MENINGITIS

Hubert L. Ried, M.D.

Recently, great interest has been aroused in the occurrence of subdural effusions during the course of the bacterial meningitides. McKay, Ingraham and Matson[1] in 1950 reported a series of twenty-three patients treated for Type B influenzal meningitis. In this group, eight were found to have collections of subdural fluid. Smith, et al.[2] have recently summarized the incidence of subdural effusions in forty-three patients treated for various types of meningitis. In their group, twenty were found to have subdural effusions as a complication of the meningitis. Subdural effusions occurring in three children during the course of meningitis were seen at Children's Hospital, Washington, D. C.

Case 1 M. H. 50-11366

M. H., a six month old colored male, was admitted on October 2, 1950 with a history of an upper respiratory infection for three days. The day prior to admission, the infant developed fever, lethargy, and vomiting. Past history was non-contributory. Birth and developmental history was normal. Feeding history was not unusual and family history revealed no pertinent details.

Physical examination disclosed a well developed and well nourished colored male infant who appeared acutely ill and lethargic. The temperature was 103.8, pulse 180 per minute, and respirations 40 per minute. The anterior fontanel was open 3 x 3 centimeters and was full but not bulging. There was no nuchal rigidity. Moderate hyperemia of the nasal mucosa with a slight mucopurulent nasal discharge was observed. The pharynx and tonsils appeared slightly inflamed. The neurologic examination was essentially normal except for a positive Brudzinski's sign.

The blood count on admission showed a hemoglobin of 9.9 grams. The white blood count was 12,000 cells per cubic millimeter with 50 per cent polymorphonuclears, 6 per cent band forms, and 44 per cent lymphocytes. Urinalysis was essentially normal. Spinal fluid was cloudy and contained 11,000 white cells per cubic millimeter with 98 per cent polymorphonuclears. The protein was 190 milligrams per cent and the sugar 4 milligrams per cent. *H. influenzae, Type B* was isolated from the fluid.

Treatment consisted of parenteral sodium sulfadiazine, aureomycin intravenously, and *H. influenzae, Type B* antiserum intramuscularly. Aqueous penicillin, parenteral fluids, and blood transfusions were administered during the acute stage.

An initial improvement occurred, but approximately 20 hours after admission, generalized convulsive episodes were noted. These were treated with barbiturates. Thereafter, the convulsive seizures recurred intermittently and the temperature fluctuated between 100.0 and 102.0. Spinal puncture 48 hours after admission showed a decrease in the cell count and protein with a rise in the sugar content. However, culture was still positive.

Chloramphenicol (Intramuscular) supplied by Parke Davis Co., Detroit, Mich. for inustigational use.

Case 1: M. H., 50-11366. From the ward service of Dr. Edward Lewis
Case 2: J. G. A., 51-2333. From the ward service of Dr. William Anderson
Case 3: R. B., 51-1815. From the private service of Dr. Stanley Steinberg

On the fourth hospital day streptomycin was substituted for aureomycin. Clinically the child's condition remained unchanged. On this day bilateral subdural punctures were done. Approximately 15 milliliters of viscid, bloody fluid was withdrawn from the right side. There was no marked improvement and intermittent convulsions persisted. On the seventh hospital day the subdural space was again entered and 30 milliliters of xanthochromic fluid was obtained from the right subdural space. Thereafter, daily subdural punctures were performed and on each occasion fluid was obtained from the right side. During this period, the child became more alert and the convulsions subsided. On the tenth hospital day bilateral temporal burr holes were done. At operation, a moderate amount of xanthochromic, blood-tinged fluid escaped from the left side when the dura was opened. There was no evidence of a membrane. On the right side no free fluid was encountered when the dura was opened and no membrane was noted. The infant's postoperative course was uneventful. Within forty-eight hours after surgery the temperature returned to normal. There were no further convulsions and the child improved remarkably. All medications were discontinued on the eighteenth hospital day and the child was discharged twenty-four days after admission.

Since discharge from the hospital the child has been doing well and has remained asymptomatic. There has been no increase in head circumference.

Case 2 J. G. 51-2333

J. G., a seven month old white male infant was admitted on February 23, 1951 with a history of fever of three days' duration. There were no other symptoms. The fever had not responded to oral penicillin therapy. The child became irritable and hospitalization was advised.

Past history was not remarkable. Birth and development had been normal. Family history revealed no pertinent details.

Physical examination revealed a well developed and fairly well nourished white male appearing acutely ill. The temperature was 103.4, pulse 130 per minute and respirations 32 per minute. The anterior fontanel was open 2 x 2 centimeters and was full with a slight increase in tension. There was no nuchal rigidity. Both ear drums were moderately injected and the landmarks of the left drum were obliterated. There was some tenderness over both mastoid areas. The pharynx and tonsils were slightly injected. Remaining physical examination was negative. There were no petechiae seen. The only abnormal neurologic finding present was a positive Brudzinski's sign.

Blood count on admission revealed 10 grams of hemoglobin, 3,900,000 red blood cells, 27,000 white blood cells per cubic millimeter with 38 per cent segmented forms, 15 per cent band forms, and 48 per cent lymphocytes. Urinalysis was essentially normal. Spinal fluid was cloudy and contained 11,475 white blood cells with 93 per cent lymphocytes and 7 per cent polymorphonuclears. Many gram negative diplocci were seen on smear and culture yielded *Neisseria meningitidis*. Blood culture was sterile. Nasopharyngeal and throat cultures failed to grow the causative organism. A roentgenogram of the mastoid regions was normal.

Treatment consisted of intramuscular penicillin, parenteral sulfadiazine, and intramuscular chloramphenicol. On the fourth hospital day, oral chloramphenicol was substituted for the intramuscular drug. Parenteral fluids and blood transfusions were administered. An initial response was obtained but on the second hospital day the temperature rose to 101.6. Bilateral subdural punctures were performed on the fourth hospital day. Eight milliliters and 5 milliliters of xanthochromic fluid was obtained from the left and right sides respectively. Thereafter, subdural punctures were performed daily and xanthochromic fluid in diminishing amounts was obtained.

These became negative on the twelfth hospital day. Spinal taps were done during this interim and showed marked improvement.

A concomitant clinical improvement was noted. All medications were discontinued on the fourteenth hospital day, and the infant was discharged seventeen days after admission. He was seen two weeks later, and was asymptomatic.

Case 3 R. B. 51-1815

R. B., a five month old white male was admitted on February 11, 1951 with a history of lethargy, fever, and anorexia. Past history revealed physiologic jaundice at birth. Feeding and family histories were non-contributory.

Physical examination revealed a well developed, well nourished, critically ill white male who appeared extremely lethargic but responded to nocioceptive and proprioceptive stimuli. The temperature was 103.8, the pulse 140 per minute, and respirations 38 per minute. The anterior fontanel was open 2 x 3 centimeters, slightly bulging, and tense. Definite stiffness of the neck and back were present. There were occasional jerking movements of the arms and legs. The remaining physical examination was normal. Neurological examination revealed hyperactive but equal deep tendon reflexes. Positive Kernig's and Brudzinski's signs were present.

Initial blood count showed 9 grams of hemoglobin, 3,700,000 red blood cells per cubic millimeter, 3,350 white blood cells per cubic millimeter, with 70 per cent lymphocytes, 6 per cent segmented forms, 8 per cent band forms and 16 per cent young forms. Urinalysis was normal. A lumbar puncture yielded cloudy fluid with 485 white blood cells per cubic millimeter, 91 per cent of these being polymorphonucleous and 9 per cent lymphocytes. Smear revealed gram negative diplococci. Cultures of spinal fluid and blood yielded *N. meningitidis*. Nasopharyngeal smear cultured normal flora.

Treatment consisted of intravenous fluids and blood transfusion, intramuscular chloramphenicol and parenteral sulfadiazine.

An initial improvement was obtained but on the second hospital day, a secondary and persistent rise of temperature occurred and the neurological symptoms continued. Culture of the spinal fluid was sterile. On the fourth hospital day, bilateral subdural punctures were performed and yielded 10 milliliters and 20 milliliters of thick xanthochromic from the right and left sides respectively. An increasing amount of fluid was obtained from the left side, while on the right, subdural puncture soon became negative. On the sixth hospital day the temperature returned to normal, and all medications were discontinued on the eleventh hospital day. On February 28, 1951 a left craniotomy was performed and a large subdural hygroma with a definite membrane was removed. Postoperatively, the child's course was uneventful. At the time of discharge, on March 8, 1951, the infant appeared alert, playful, and neurologic examination was normal.

DISCUSSION

Despite the improved prognosis in purulent meningitis, a significant number of patients have permanent neurologic sequellae. It may be that some of these are due to subdural effusions. Our three cases showed an initial clinical and chemical improvement. This initial improvement was not sustained and subdural punctures were performed. Following repeated punctures, clinical improvement occurred. Whether or not these children would have escaped permanent sequellae had not the subdural effusions been drained will remain an unanswered question. Clinically these children

appeared to be good candidates for permanent sequellae. From the previously published reports, the incidence of subdural effusions complicating the course of purulent meningitis in children under two years of age is between 30 and 50%. Our patients were under seven months of age. As yet, the incidence of subdural effusion in older children has not been determined. Such an investigation would be worthwhile.

To date, most cases of meningitis complicated by the accumulation of subdural fluid have been associated with *H. influenzae* infection. However, similar effusions have been found in meningitis caused by *Diplococcus pneumoniae*, *Pseudomonas aeruginosa*, and *Paracolon bacillus*. Smith et al.[2] reported only one case of menigococcal meningitis associated with this entity. In this report, two patients had a meningococcal infection and one had an influenzal meningitis. From this evidence, one can conclude that subdural effusions may be associated with any of the purulent meningitides.

The mechanism responsible for the accumulation of subdural fluid in patients with meningitis is unknown. McKay et al.[1] postulate that one or several factors may play an important role. The fluid may represent either an "exudation or hemorrhage as a complication of the meningitis or as a complication of the antibiotic therapy." It is improbable that the trauma associated with subdural puncture is responsible for the effusion, because the initial examination is usually positive. Too, it is unlikely that a pre-existing subdural hematoma is present in such a high percentage of patients. Smith et al.[2] postulate that the acute inflammation in the subarachnoid space extends to the pia arachnoid with a resultant increase in permeability of the pia and an escape of fluid into the subdural space. The high protein and cell count of the accumulated fluid is responsible for an elevated osmotic pressure which draws more fluid into the space. A similar mechanism is believed to operate in the formation of subdural hematomas.[3] Membrane formation probably depends to a great extent on the protein content of the subdural fluid as well as on the increased intracranial pressure which hinders the resorption of the subdural fluid by compressing the capillary plexus of the dura[2].

In most instances the subdural fluid is found bilaterally although at the time of the initial subdural puncture, fluid may be recovered from only one side. In our three cases bilateral fluid was found initially. However, in all cases, after repeated taps, there was a tendency for fluid to disappear from one side sooner than from the other. It is, therefore, imperative that bilateral subdural punctures be performed in suspected cases.

The amount of effusion varied from 8 milliliters to 60 milliliters. The fluid was usually blood-tinged. However, after centrifuging, the supernatent portion was usually xanthochromic. Analysis invariably revealed a high protein content which ranged from 108 milligrams per cent to 1300 milligrams per cent. In two cases the spinal fluid was examined at the

time of the subdural puncture. In both instances there was a much lower protein content noted in the spinal fluid. The protein content of the subdural fluid becomes elevated on successive taps. The cellular content could not be accurately interpreted, since in most cases the fluid contained blood. The sugar content ranged from 6 milligrams per cent to 45 milligrams per cent. In all instances, smears and cultures of the subdural fluid were negative.

A combination of symptoms and physical findings prompted the investigation of the subdural spaces. In our three cases definite indications were present for the subdural puncture even though there was improvement in the spinal fluid findings. These included: continued fever, nuchal and back rigidity, bulging fontanel, and convulsions despite adequate therapy for forty-eight to seventy-two hours. In case No. 2, the indications for subdural puncture were minimal; however, fluid was obtained bilaterally. It is suggested that subdural taps be performed on any patient whose clinical course is unsatisfactory, or in whom persistence of fever and/or positive spinal fluid culture is noted after forty-eight to seventy-two hours of adequate therapy. In addition, convulsions, persistent vomiting, focal neurologic signs, or enlargement of the head serve as further indications for this procedure.

In our three cases, the subdural effusions were all discovered within the first five days of the disease. The early discovery can be attributed primarily to a high index of suspicion that such effusions occur.

In cases No. 1 and No. 3 the treatment of the subdural effusions consisted of daily puncture and ultimate surgery. When bilateral temporal burr holes were performed in case No. 1 no membrane was found. However, xanthochromic fluid was encountered on the left side when the dura was opened. In case No. 3 a definite membrane with enclosed xanthochromic fluid was found on the left side. Case No. 2 was not operated upon as the amount of subdural fluid recovered on repeated taps decreased rapidly and no further untoward symptoms were encountered. McKay et al.[1] performed bilateral temporal burr holes on nine cases of subdural effusions associated with *H. Influenza* meningitis. In six patients a membrane with encapsulated fluid was encountered and excised. In twenty patients reported by Smith et al.[2] only two were treated surgically. One of these was found to have membrane encapsulated fluid. At present it would seem best to reserve surgery for those cases of meningitis with subdural effusions which yield repeated large amounts of subdural fluid.

SUMMARY

Three cases of subdural effusions complicating bacterial meningitis are presented. In two cases, *Neisseria meningitidis* was isolated as the causa-

tive agent. *H. influenzae, Type B.* was recovered from the spinal fluid of the third patient. All three patients were infants under seven months of age.

Findings which suggest the accumulation of subdural fluid in cases of meningitis include: unsatisfactory clinical course after forty-eight to seventy-two hours of adequate therapy as evidenced by continued fever, persistent positive spinal fluid culture, recurrent vomiting, bulging fontanel, nuchal rigidity, convulsions, focal neurologic signs and enlargement of the head. It is suggested that bilateral subdural taps be performed on all children whose course during treatment is complicated by any of the above noted findings.

In the cases reported, bilateral subdural effusions were discovered within the first five days of the disease. The subdural fluid was usually blood-tinged and xanthochromic and ranged from 10 milliliters to 60 milliliters. The protein content ranged from 108 milligrams per cent to 1300 milligrams per cent. In all instances the subdural fluid was sterile on culture.

Treatment consisted of repeated subdural taps in all three children and craniotomy in two patients. Membrane encapsulated fluid on the left side was found at time of surgery in one case. Xanthochromic fluid without membrane formation was found on the left side in the second patient. Treatment of the third patient consisted of repeated subdural punctures with gradual disappearance of the fluid. At the time of this report all patients are in good health and asymptomatic. It is suggested that therapy consist of repeated subdural punctures and that surgical intervention be reserved for those patients in whom daily large accumulations of fluid are encountered.

ADDENDUM

Since the preparation of this report, two additional patients with a subdural effusion complicating purulent meningitis have been seen at this hospital. The causative agent of the meningitis in one instance was *H. influenzae* and in the other is as yet undetermined.

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CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.

Assisted by: Sanford Leikin, M.D.
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By Invitation: John A. Washington, M.D.

Sanford Leikin, M.D.

This two year old white male was admitted to this hospital on February 9, 1951, and died on February 11, 1951 at 1:07 P.M. The acute illness which brought this patient to the hospital on this final admission was a cold of three days' duration with fever which did not respond to aureomycin.

The patient was a product of a full term pregnancy and normal delivery. His birth weight was 6 pounds and 8 ounces. In the neonatal period he had rectal bleeding. At that time his prothrombin was 26 per cent of normal, and he received 10 milligrams of Vitamin K intravenously and a small transfusion via the umbilical vein. He recovered uneventfully. In April of 1949, he had a severe cold, following which he became anemic and acidotic. He was admitted to the hospital and treated with parenteral fluids and made an uneventful recovery. Following this, however, his respirations remained somewhat rapid, and in November he began losing weight and became pale and irritable. He was readmitted to the hospital for transfusions, at which time his urine showed some albumin. He did moderately well until shortly after Christmas, when he again became pale, weak and anorexic, with rapid respirations. He was readmitted early in January of 1950 at which time his non-protein nitrogen was 185 milligrams per cent, his carbon dioxide combining power was low, and the ratio of calcium to phosphorus was inverted. He had a bout of tetany, for which calcium gluconate was given. Intravenous pyelograms were done but the kidneys could not be outlined.

In February, 1950, the patient was admitted to another hospital, where he was studied for his illness. At that time his blood pressure was 92/45 and he had an anemia and a leukocytosis. The specific gravity of his urine varied between 1.012 to 1.007. He showed from a trace to 1 plus albumin and occasional white blood cells, but no red blood cells. Two Sulkowitch tests done on the urine were negative. A phthalein excretion test showed less than 1 per cent excretion in 120 minutes. Examination of his blood showed an azotemia of 192 milligrams per cent of non-protein nitrogen with a reversal of the calcium and phosphorus, the calcium being 6.3 milligrams per cent and the phosphorus, 10.8 milligrams per cent. His alkaline phosphatase activity was 12.8 units. His carbon dioxide combining power was 9.9 milliequivalents per liter. His sodium was 137 milliequivalents per liter.

X-ray examination of the long bones showed coarse osteoporosis with some irregularity of the epiphyseal plate. An intravenous pyelogram showed that the kidneys could not be visualized after the injection of the dye. This was interpreted as being due to a very poor power of concentration. A cystogram showed the bladder to be normal in size and shape with no reflux of the dye into the ureters.

Because of the inability to see dye in the kidney, a bilateral retrograde pyelogram was done. The kidney outline on the left was fairly well visualized. It appeared quite small and measured roughly 4 centimeters in its greatest diameter. The outline on the right was not seen. The calyces were not well filled on either side. The left

pelvis was definitely smaller than normal, while the right was grossly within normal limits. The ureters were normal. The impression was a hypoplastic left kidney, and a probable hypoplastic right kidney.

The patient was discharged on calcium lactate and ferric ammonium citrate with a feeding schedule providing 40 ounces of fluid a day.

In December of 1950, the patient was admitted to this hospital for study and transfusion. His hemoglobin at that time was 9.5 grams and his red blood count was 2.6 million. His carbon dioxide combining power was 21 volumes per cent, non-protein nitrogen was 169 milligrams per cent. He was given a blood transfusion and discharged.

His developmental history was normal except that, although he could stand, he was not able to walk. There was no history of tuberculosis, diabetes, kidney disease, asthma or hay fever in the family.

Physical examination on admission showed that his anterior fontanel admitted one finger. His pharynx was slightly injected.

There was grade 2 systolic murmur heard to the left of his sternum and at the apex and loud ronchi were heard throughout both lung fields. The child was somewhat pale and he was slightly dehydrated.

His hemogram showed an anemia with 2 million red blood cells, and a hemoglobin of 8 grams. He had a mild leukocytosis of 14,500 cells. His urine showed 100 milligrams of albumin with a 2 plus acetone and few coarse granular casts. The carbon dioxide combining power of the blood was 19 volumes per cent.

The patient was treated with blood and $\frac{1}{6}$ molar lactate solution to correct his acidosis. On February 11th, his condition was noted to be very poor, his respirations were labored and his heart rate was rapid. He was markedly dehydrated. A carbon dioxide combining power of the blood done at this time was 13 volumes per cent. At 1:07 P.M. of the same day the patient suddenly stopped breathing and no heart action could be detected. In spite of stimulants the patient expired. An autopsy was performed.

DISCUSSION

John A. Washington, M.D.: It is apparent that this baby suffered from chronic renal failure. This was demonstrable soon after he was one year of age when the non-protein nitrogen was found to be 185 milligrams per cent. It is reasonable to suspect that renal failure existed at six months of age when he was described as being acidotic during a severe cold. The thorough investigation done in February, 1950, when he was about fifteen months old included a retrograde pyelogram which gave the impression that the kidneys were hypoplastic. Presumably this condition dated from birth. Severe impairment of kidney function was further demonstrated by specific gravity readings and a phthalein excretion test.

My interest centers upon the high serum phosphorus and the X-rays of the long bones showing coarse osteoporosis with some irregularity of the epiphyseal plate. It seems likely that some renal rickets was present although no deformities were noted clinically. Probably deformities would not occur in so young a child. As has been pointed out by Dr. E. A. Park and others, renal rickets is not rickets such as occurs from lack of Vitamin

D but rather a demineralization of bone probably due to hyperparathyroidism. The latter condition occurs in the presence of prolonged renal failure as the result of the inability of impaired kidneys to excrete phosphorus. Retention of phosphorus in the blood stream stimulates the activity of the parathyroid glands, the function of which appears to be the regulation of phosphate excretion. In this case it seems likely that the retention of phosphorus had persisted long enough to cause hypertrophy of the parathyroid glands and x-ray changes in the long bones, actually in the nature of osteitis fibrosa, such as is seen in hyperparathyroidism.

The findings of low serum calcium and of high serum phosphorus is not a constant one in cases of renal rickets, but is entirely consistent with that diagnosis. Normal blood pressures in the presence of chronic renal failure also are not unusual.

In summary I would expect the autopsy to show congenital hypoplasia of the kidneys and in addition, some hypertrophy of the parathyroid glands in response to prolonged phosphorus retention.

PATHOLOGIC DISCUSSION

David F. Bell, M.D.: The pertinent pathological findings at postmortem examination were seen in the kidneys; the right kidney weighed 11 grams and the left kidney weighed 8 grams (normal for this age would be 47 grams and 46 grams respectively). The kidneys cut with increased resistance and the capsules stripped with difficulty revealing granular gray and mottled red cortices. The cortices averaged less than 0.2 centimeters in thickness and the architecture was distorted by an increased amount of fibrous tissue. The mucosa of the ureters was slightly roughened and pale. Both ureters were patent throughout, as was the urethra.

Microscopically, the kidneys presented the typical picture of a chronic pyelonephritis.

The anatomical diagnoses in this patient were:

1. Hypoplasia of the kidneys secondary to chronic pyelonephritis
2. Acute bronchitis
3. Myocardial hypertrophy
4. Fatty metamorphosis of the liver
5. Rickets.

We wish to thank Dr. Washington for his very complete clinico-pathologic correlation of this case in his discussion. There is very little that can be added in this respect.

However, the underlying cause of the renal hypoplasia microscopically appears to be one of infection, rather than a congenital malformation. The identical clinical and laboratory picture has resulted.

An excellent study has been published recently* in regard to parathyroid hyperplasia in chronic renal disease which is well worth review by those interested in the interrelationship between the parathyroid glands and the kidney. In this particular patient the parathyroid glands were slightly enlarged, but microscopically the cellular component was not that usually described in parathyroid hyperplasia.

* Fritz, G. E. and Brines, O. A.: The Cell Type of Secondary Parathyroid Hyperplasia, Am. J. Path. 27:2 (March–April) 1951.

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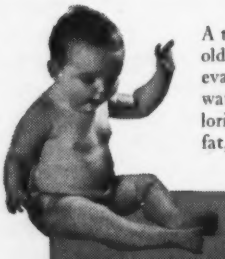

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